Aerobiology and Secondary Infection

by
Clayton G. Loosli, M. D.
Assistant Professor of Medicine
University of Chicago

Intramural aerobiology has become increasingly important as a result of accumulating evidence which indicates that air may be the chief vehicle for the dissemination of pathogenic bacteria which contaminate wounds and burns, and which cause primary and secondary infections of the respiratory tract. In World War I delayed deaths among gas casualties were due principally to secondary infection of the lungs. Among the gassed victims, who did not succumb, infection is considered to have played an important role in producing the residual changes in the lungs such as bronchitis, emphysema, and fibrosis. Likewise the great majority of deaths following influenza and measles during the last war were due to secondary bacterial infections of the lungs.

The pathological reaction elicited by the irritant gases, especially mustard gas, in the pulmonary tract is similar in many respects to that described in the lungs of cases dying of measles and, especially, of influenza. These changes render the victims highly susceptible to infections with respiratory pathogens. The protective lining of the air passages and the eliminative mechanisms of the lungs are destroyed by the viral and chemical agents. The necrotized epithelial cells which once lined the trachea and bronchi and the edema fluid which oozes into the air passages from the underlying swollen connective tissue provides an excellent culture medium and fertile field for growth of organisms. In these cases pneumonia inevitably follows the aspiration or inhalation of infected exudate containing pathogenic bacteria from the upper air passages into the already irritated and congested lung parenchyma.

The bacterial flora found in the lungs of cases dying of pneumonia following influenza and gas poisoning is similar to that found in the nose and throat. Usually one of the pathogenic organisms associated with upper respiratory tract infections were recovered. In some camps hemolytic streptococci predominated while in others influenza bacillus, pneumococci, and staphylocci were recovered in the majority of cases. Occasionally Friedlanders bacillus was found. In the lungs of gassed victims dying of secondary pneumonia, on the other hand, hemolytic streptococci, and pneumococci were rarely recovered. Predominantly non-hemolytic streptococci and other organisms considered non-pathogenic and normal inhabitants of the nose and throat were present. The pathogenic organisms causing the secondary pulmonary infections in influenza and measles patients were generally considered to have been already present in the noses or throats before

they contracted their primary disease, or were acquired by inhalation of infectious droplets from direct or close contact with infected individuals or carriers among the doctors, nurses, attendants and fellow patients. In many cases, however, the organisms were acquired after admission to clean hospital wards under strict isolation precautions. This was also true in cases of secondary infections of fracture wounds and presumably also occurred in some cases of delayed secondary pneumonia following gassing.

The spread of respiratory disease by inhalation of infectious droplets was generally considered to occur only as a result of close contact. In the past decade, however, air contaminated with pathogenic bacteria and viruses has been shown to be the spread of certain diseases. Recent investigations have shown that in sneezing, coughing and talking both well and ill persons expel into the air large numbers of saliva droplets containing microorganisms. The smaller droplets dry and remain suspended in the air as droplet nuclei. The larger ones fall to the floor, evaporate and may become resuspended in the air as dust. These droplet nuclei and dust particles containing bacteria or virus may be conveyed through the air for long distances. Dried saliva particles which float in the air from individuals with respiratory disease may contain pathogens and therefore constitute a potential source of infection. The bacterial content of the air in schools, hospital wards and institutions is roughly proportional to the degree of occupancy and activity. The number of pathogenic bacteria in the air are in general proportional to the number of respiratory infections among the occupants, but are small compared to the total air flora. Such pathogens may, however, remain viable in the dust for long periods of time.

Although actual proof of aerial spread of infection is difficult to establish, clinical and bacterial studies made in an effort to control cross infection in hospitals indicate that droplet nuclei and infectious dust particles may be of special importance in the spread of viral agents and bacteria such as streptococci, diphtheria bacilli and staphylococci. Spread of measles and chicken pox have been shown to occur under conditions where direct and indirect contact was excluded. In general the spread of these diseases followed the flow of air currents. Hemolytic streptococcal infection outbreaks in hospitals have been described where the disease spread from one floor to another in the direction of air currents. Relapses and complications in scarlet fever patients in wards have been shown to be due to reinfections with different types of streptococci. Such types were recovered from the ward dust and air. A case of streptococcus infection has been reported in a ward attendant who swept out a room five days following the discharge of a patient who had a hemolytic streptococcus

infection of the same type. Likewise, cross infections in diphtheria wards were shown to be due to reinfection with another type of diphtheria bacillus. Similar types were recovered from the floor dust in large numbers at the time the cross infections occurred.

Rapid increase in secondary streptococcal infections of burns after patients were admitted to the hospital were found to be due to cross infections from cases already present. Organisms recovered from the air and dust were similar to those causing the complications. Likewise it has been shown that many hemolytic streptococcus infected war wounds occurred after admission to the hospital and were cross infections from types of streptococci present in the air and dust of the wards. The rough handling of dry dressings from infected wounds and burns results in the liberations of many organisms into the air. The presence of large numbers of viable and virulent hemolytic streptococci have been demonstrated on blankets and dust in the wards containing streptococcus infected patients. The sweeping of the floor, changing bed clothes, and making beds in the infected patients rooms causes a marked increase in the bacterial content of the air. Such dust particles containing pathogens may remain and float on blanket lint for long periods. Many thousands of pathogenic organisms may be present in one gram of dust. In studies of simulated room environments viable and virulent hemolytic streptococci have been recovered several weeks after introduction into room air.

Direct evidence of the aerial spread of disease can be obtained only under well controlled experimental conditions. Although the spread of influenza during the 1918 epidemic was considered to have been transmitted most likely by droplet infection, actual proof was lacking. Since the isolation of the influenza virus, however, clinical influenza has been produced in human volunteers following inhalation of air, containing virus. Aerial transmissions in ferrets and mice can be readily demonstrated by merely exposing these animals to atmospheres into which influenza virus has been sprayed as fine droplets. Under certain conditions the influenza virus may remain in room air retaining its potency to infect and kill mice placed in the rooms several hours after its introduction into the air. Mice normally are not susceptible to streptococci and pneumococci sprayed in the air in relatively large numbers. However, when given a sublethal influenza A infection they become highly susceptible to secondary bacterial infection and die of extensive pneumonia when allowed to breathe the bacteria-laden air.

The above clinical, bacteriological and experimental evidence shows that if secondary infections of various types are to be prevented, the bacterial and viral content of the air in which

susceptible to respiratory tract infection from the inhalation of air-borne pathogens as are others whose open wounds and burns become so readily infected from contaminated air. In preventing secondary infections, it is important to recognize that the infective agent may be conveyed through the air as droplets, droplet nuclei or dust particles. All means should be applied which will keep the bacterial content in the air below the infective level. The following are some of the more important recommendations of numerous investigators for the control of secondary infections.

Measures which help to control droplet infection:

1. Segregation and if possible isolation of all patients.
2. All attendants, doctors, nurses, orderlies and visitors should be adequately masked (Canton flannel) and gowned.
3. Attendants should be free from upper respiratory infections or grippe, sore throat or common cold.
4. Adequate spacing of beds with barriers between beds.
5. Isolation and prompt treatment with sulfonamide therapy of patients who contract a secondary infection with determination of etiological agent when possible.
6. Masking of patients during ward cleaning and bed making.

Measures designed to control the dispersal of dust borne particles and droplet nuclei in the air:

1. Oiling of floors.
2. Sweeping with oiled or moistened brooms and bactericidal sweeping compounds.
3. Treating blankets and bed clothes with oil preparations, bactericidal compounds.
4. Careful handling and disposal of infected dressings.
5. Adequate ventilation with sufficient air changes to prevent the building up of high bacterial concentration in the air.

Measures designed to sterilize the room air:

1. Adequately lighted (daylight) wards and rooms.
2. Ultraviolet radiation.
3. Germicidal agents employed as mists or aerosals.

    a. Propylene glycol, one part in from 2 to 10 million parts of air.
    b. Triethylene glycol in one to two hundred million parts of air.

Under stress of emergency many of the above principles and

measures designed to prevent and control secondary infections following gassing may not be practical. As many as possible, however, should be tried if good results are to be attained.

Because of the high susceptibility of gassed patients to secondary infections of the pulmonary tract, careful clinical control should be exercised in their management. Daily observations should be made to detect early any pneumonias which may develop. Fever, cough, increased respiratory and pulse rate and leucocytosis indicate a pulmonary infection. If possible the patient should be placed under strict isolation and prompt treatment with sulfonamides instituted. If laboratory facilities are available the etiological agents causing the pneumonia should be ascertained as an aid to prognosis and drug treatment. In secondary pneumonias following exposure to pulmonary irritants, the flora is often mixed and therefore a drug with the wide range of effectiveness which produces the fewest complications should be used. Sulfadiazine is now generally recommended as the first choice in the treatment of pneumonia where the etiological agents are not determined, as well as in those cases caused by hemolytic streptococci, pneumococci, staphylococci, influenza bacillus or Friedlanders bacillus. The dosage of drug recommended is the same as that used in the treatment of the primary pneumonias. Four grams is given as the initial dose followed by one gram every four hours until seventy-two hours after the temperature has returned to normal. Whether the recommended schedule of treatment should be continued would depend on the clinical course of the patient. Blood drug levels should be determined where possible to be sure of adequate therapy. Where sulfadiazine is not available sulfathiazole is the next drug of choice with the third choice being sulfapyridine. Regardless of the drug used the dosage is the same.

Whether the sulfonamide drugs should be given prophylactically as a pneumonia preventative to gassed victims is problematical. It would seem advisable in case of a hemolytic streptococcus throat or pneumonia outbreak in a ward. However, if careful clinical observations are made the secondary pneumonias in these cases can be detected early enough to bring about favorable results with sulfonamide treatment after the diagnosis is made. If a sulfonamide drug is to be used prophylactically the dosage should be essentially the same as used during treatment: four grams as an initial dose followed by one gram every six hours.

When using sulfonamide drugs in the treatment of pulmonary infections the possible complications should be kept clearly in mind. Adequate fluid out-put (over 1000 cc. a day) is highly essential to avoid renal complications. The sulfonamides should not be considered as a substitute for other accepted procedures in the treatment of complications such as drainage of abscesses and emphysema cavities. General medical measures should not be neglected in treatment of these pneumonias with sulfonamide drugs.